POTASSIUM EFFECTS ON THE ELECTROCARDIOGRAM OF THYROID DEFICIENCY

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As there is no method for detecting and measuring circulating thyroid hormone, indirect investigations have to be used for the accurate diagnosis of thyroid deficiency. It is difficult to obtain evidence that thyroid deficiency in man is complete. In addition to the ordinary metabolic and biochemical findings, the response to injected pituitary thyrotrophic hormone must be negative (Sharpey-Schafer and Schrire, 1939), histological changes are present in the pituitary gland, and there is possibly an excess of thyrotrophic principle in the circulating blood (Collard, Mills, Rundle, and Sharpey-Schafer, 1940). Clinical observations may be quite indecisive. The cardiogram, however, affords a valuable measure. Elsewhere, evidence will be given that when thyroid deficiency is complete, reversible cardiographic changes are always present. The changes show a constant picture: in addition to a general low voltage, the T waves are flat or in a few cases inverted. It can usually be proved that thyroid deficiency is not complete in cases that do not show a typical cardiogram, though the presence of a classical one does not necessarily imply that the patient is strictly comparable to a thyroidectomised animal. The action of thyroid hormone on such cardiograms is well known. Provided a sufficient dosage is given, the appearances return to those which are normal for the individual. Of special interest are those rarer cases with T wave inversion. Thyroid causes such T waves to become upright, yet their presence is sometimes mistaken for conditions such as coronary arterial disease. Any other method of altering the cardiogram of thyroid deficiency might throw some light on its mechanism and prove of value in the differential diagnosis of low voltage curves generally. This paper reports the effect on such cardiograms of raising the serum potassium.

MATERIAL

The procedure is reported elsewhere (Sharpey-Schafer, 1943). Many patients showed a later rise of serum potassium than others without thyroid deficiency, possibly due to a slower rate of absorption from the intestine. The evidence for thyroid deficiency in 12 patients is given in the Table. Signs and symptoms are those given in the *Report on Myxœdema*, 1888, a description that has not been bettered. The measurement of response to thyrotrophic hormone has been previously published (Sharpey-Schafer and Schrire, 1939). Many cases received very large doses of pituitary extract. A biopsy of the thyroid is available in Case 1, and post-mortem material in the untreated state in Case 7. There was clear clinical and other evidence in Case 4 that thyroid deficiency was not complete; the cardiographic changes were, however, similar to those found in cases of proved complete deficiency. The patient with Addisonian anæmia (see Fig. 3) on the occasion of the observation had 1,100,000 red cells per c.mm. and 24 per cent hæmoglobin. The venous pressure was raised, and pitting œdema was present.

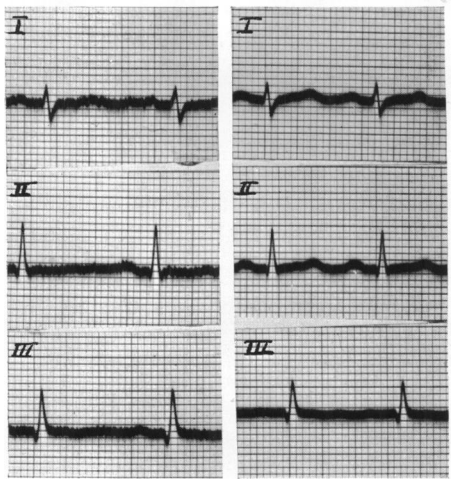
TABLE

SUMMARY OF GENERAL FEATURES AND OF SIZE OF T WAVES BEFORE AND AFTER TREATMENT

Case	Age Sex	Symptoms and signs	B.M.R. per cent	Cholesterol mg. per 100 c.c.	Response to thyrotrophic hormone	Size of T waves in electrocardiogram (millivolts)					
						Untreated		After potassium		After treatment with thyroid	
						Upright	Flat or Inverted	Upright	Flat or Inverted	Upright	Flat or Inverted
1	F. 20	+	−26	150–315	0	I 0·05	II 0·00 III 0·00	I 0·10 II 0·10	III 0·00	I 0·13 II 0·23 III 0·05	
2	F. 70	+	−34	254–353	0		I 0·00 II 0·05 III 0·10	I 0·05 II 0·04	III 0·00	I 0·12 II 0·20 III 0·10	
3	F. 40	+	−26	150–290	0		I 0·10 II 0·12 III 0·05	I 0·10 II 0·13	III 0·00	I 0·20 II 0·20	III 0·00
4	F. 49		−31 to −37	335–400	sl.+		I 0·05 II 0·10 III 0·15	I 0·10 II 0·05	III 0·05	I 0·15 II 0·40 III 0·25	
5	F. 10	+	−27	415	0	I 0·03 II 0·07 III 0·02		I 0·08 II 0·20 III 0·10		I 0·15 II 0·22 III 0·10	
6	F. 48	+	−36 to −46	180	0		I 0·00 II 0·00 III 0·00	I 0·02 II 0·10 III 0·05		I 0·13 II 0·20 III 0·10	
7	F. 63	+	−41	270–290	0	II 0·10 III 0·10	I 0·00	I 0·10 II 0·20 III 0·13		I 0·10 II 0·20 III 0·13	
8	F. 58	+	−32	217	0	I 0·02	II 0·00 III 0·05	I 0·20 II 0·15	III 0·05	I 0·30 II 0·30	III 0·00
9	F. 59	+	−15	184	0	III 0·05	I 0·05 II 0·00	II 0·10 III 0·13	I 0·00	I 0·15 II 0·15	III 0·00
10	F. 66	+	−34	296	0		I 0·00 II 0·00 III 0·00	I 0·10 II 0·20 III 0·05			
11	F. 66	+	−32	270	0		I 0·00 II 0·00 III 0·00	I 0·04 II 0·10 III 0·05			
12	F. 41	+	−32	270–310	0		I 0·00 II 0·00 III 0·00	I 0·02 II 0·07 III 0·05		I 0·10 II 0·15 III 0·05	

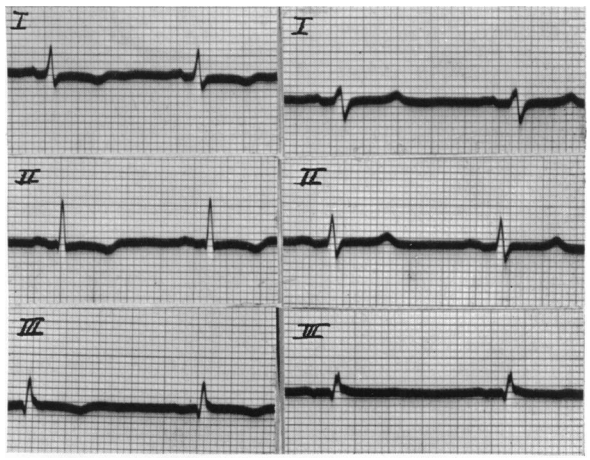
RESULTS

The results are shown in the Table. T wave voltage after treatment with thyroid is given in the last column. All cases showed the same change to a greater or lesser degree. Those with flat T waves showed a rise of the T wave after potassium (Fig. 1). If the T waves were inverted, they also became upright (Fig. 2). The case with Addison's anæmia (Fig. 3) showed a different change. The control cardiogram cannot be distinguished from one due to thyroid



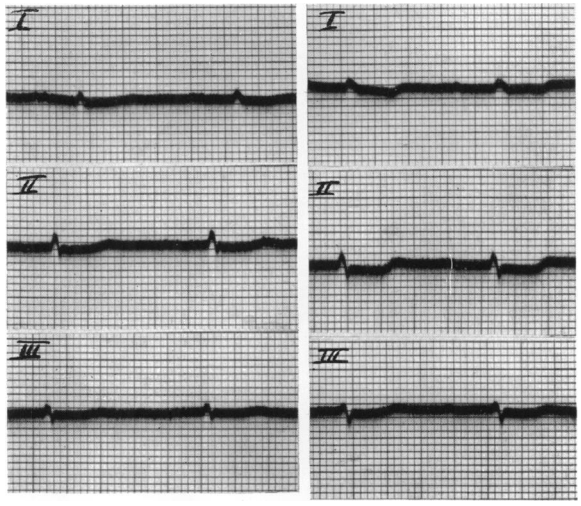
A FIG. 1. B

FIG. 1.—*Case* 1. *Myxœdema.* Cardiograms before and after treatment with potassium.
(A) Before .. .. .. Serum potassium=20 mg. per 100 c.c.
(B) 1½ hours after .. Serum potassium=25 mg. per 100 c.c.



A FIG. 2. B

FIG. 2.—*Case* 3. *Myxœdema.* Cardiograms before and after treatment with potassium.
(A) Before .. .. .. Serum potassium=20 mg. per 100 c.c.
(B) 2½ hours after .. Serum potassium=34 mg. per 100 c.c.



A FIG. 3. B

FIG. 3.—*Addison's Anæmia.* Cardiograms before and after treatment with potassium.
(A) Before .. .. Serum potassium=22 mg. per 100 c.c.
(B) 2½ hours after .. Serum potassium=28 mg. per 100 c.c.

deficiency, especially since the heart rate is relatively slow. After potassium S–T depression is accentuated without much change in the T wave.

DISCUSSION

The results indicate that the effect of potassium on the T wave of thyroid deficiency is similar to the effect of thyroid hormone. The time taken for action is, however, very different for the two substances. While potassium produces changes in a few hours, thyroid takes

days. It is not suggested that there is any direct connection between these two effects. The serum potassium in cases of thyroid deficiency lies within normal limits, and, although the voltage of the normal T wave may be lowered by lowering serum potassium (unpublished data), there is no evidence that cardiographic appearances in hypothyroidism are due to electrolyte changes. It is possible that potassium merely accentuates deflections that are already present, so far as S–T interval and the T wave are concerned (Sharpey-Schafer, 1943). If this is so, the results described here suggest that no fundamental change in pattern is involved in the production of the cardiogram of thyroid deficiency. Since the first report by Zondek (1918) numerous authors have written on the cardiographic findings in myxœdema. Ohler and Abramson (1934), reviewing such papers and reporting on 35 cases, discussed the following possible causes: (1) sclerosis of the coronary arteries, (2) anæmia, (3) cardiac dilatation and sluggishness, (4) vagal stimulation, (5) cutaneous resistance, and (6) pericardial effusion. They showed that there was good evidence against causes (2), (3), (4), & (5), and, since potassium further inverts the T wave of myocardial infarction, our findings support Ohler and Abramson in dismissing cause (1). Indeed, T inversion in hypothyroidism responds to potassium in the same manner as T inversion in preponderance of a ventricle. We have post-mortem evidence in untreated cases that the classical cardiogram can occur in the absence of pericardial effusion. The effect of potassium on the cardiogram of pericarditis has not yet been investigated.

Summary

The flat T wave of thyroid deficiency became upright after potassium. Inverted T waves also became upright, a response which is similar to that in preponderance of a ventricle and unlike that of myocardial infarction. A case of Addison's anæmia showed accentuation of the S–T depression.

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